

WEST Search History

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DATE: Friday, January 09, 2004

Hide?	Set Name	Query	Hit Count
		<i>DB=USPT; PLUR=YES; OP=OR</i>	
<input type="checkbox"/>	L7	L5 and screening	8
<input type="checkbox"/>	L6	L5 and neurotoxin	1
<input type="checkbox"/>	L5	C same elegans same marker same neuron	9
<input type="checkbox"/>	L4	L3 and screening	3
<input type="checkbox"/>	L3	L2 and neurotoxin	12
<input type="checkbox"/>	L2	C. elegans same marker same neuron	1947
<input type="checkbox"/>	L1	C. elegans same marker	2007

END OF SEARCH HISTORY

FILE 'HOME' ENTERED AT 15:13:48 ON 09 JAN 2004

=> file medline biosis embase scisearch caplus
COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
0.63	0.63

FULL ESTIMATED COST

FILE 'MEDLINE' ENTERED AT 15:15:42 ON 09 JAN 2004

FILE 'BIOSIS' ENTERED AT 15:15:42 ON 09 JAN 2004
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=> s C. elegans
L1 22518 C. ELEGANS

=> s l1 and fluorescent
L2 869 L1 AND FLUORESCENT

=> s l2 and (neuron or neuronal)
L3 145 L2 AND (NEURON OR NEURONAL)

=> s l3 and screen?
L4 15 L3 AND SCREEN?

=> dup rem
ENTER L# LIST OR (END):14
PROCESSING COMPLETED FOR L4
L5 15 DUP REM L4 (0 DUPLICATES REMOVED)

=> d l5 tot ibib abs

```
=> s l3 and neurotoxin
L6          7 L3 AND NEUROTOXIN

=> s l3 and neuroprotective
L7          2 L3 AND NEUROPROTECTIVE
```

```
=> dup rem
ENTER L# LIST OR (END):16, 17
PROCESSING COMPLETED FOR L6
PROCESSING COMPLETED FOR L7
L8          4 DUP REM L6, L7 (5 DUPLICATES REMOVED)

=> d l8 tot ibib abs
```

=> s l3 and dopamin?
L9 8 L3 AND DOPAMIN?

=> s l3 and dopamine
L10 7 L3 AND DOPAMINE

=> dup rem
ENTER L# LIST OR (END):l9, l10
PROCESSING COMPLETED FOR L9
PROCESSING COMPLETED FOR L10
L11 4 DUP REM L9, L10 (11 DUPLICATES REMOVED)

=> d l11 tot ibib abs

```

=> s l3 and neuron specific promoter
L12          0 L3 AND NEURON SPECIFIC PROMOTER

=> s l3 and neuronal promoter
L13          1 L3 AND NEURONAL PROMOTER

=> s l3 and CMV
L14          0 L3 AND CMV

=> s l3 and promoter
L15          37 L3 AND PROMOTER

=> dup rem
ENTER L# LIST OR (END):l15
PROCESSING COMPLETED FOR L15
L16          27 DUP REM L15 (10 DUPLICATES REMOVED)

=> s l3 and py <= 2001
      2 FILES SEARCHED...
      4 FILES SEARCHED...
L17          94 L3 AND PY <= 2001

=> dup rem
ENTER L# LIST OR (END):l17
PROCESSING COMPLETED FOR L17
L18          68 DUP REM L17 (26 DUPLICATES REMOVED)

=> d l16 tot ibib abs

```

L5 ANSWER 9 OF 15 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
 ACCESSION NUMBER: 2001:532653 BIOSIS
 DOCUMENT NUMBER: PREV200100532653
 TITLE: Modeling Parkinson's disease in *C. elegans*: Studies of neurotoxin induced DA neuron degeneration.
 AUTHOR(S): Nass, R. [Reprint author]; Hall, D. H.; Miller, D. M.; Blakely, R. D.
 CORPORATE SOURCE: Molec Neurosci and Pharm, Vanderbilt U SOM, Nashville, TN, USA
 SOURCE: Society for Neuroscience Abstracts, (2001) Vol. 27, No. 1, pp. 1145. print.
 Meeting Info.: 31st Annual Meeting of the Society for Neuroscience. San Diego, California, USA. November 10-15, 2001.
 ISSN: 0190-5295.
 DOCUMENT TYPE: Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)
 LANGUAGE: English
 ENTRY DATE: Entered STN: 14 Nov 2001
 Last Updated on STN: 23 Feb 2002

AB The dopamine transporter DAT, which is the target for many psychoactive drugs, provides the cellular gateway for the accumulation of neurotoxins that evokes **neuronal** death and Parkinson-like syndrome in animal models. We have previously cloned the *C. elegans* DAT (DAT-1), and have shown that it is functionally similar to mammalian DATs and expressed exclusively in the dopamine (DA) neurons (Jayanthi et al. 1998, Nass et al. 1999, 2001). We have also developed transgenic lines which target a dat-1 promoter-gfp fusion to all 8 DA neurons in the hermaphrodite. Exposure of the reporter line to 6-OHDA results in DA **neuronal** blebbing, soma deformation, and loss of GFP expression. Antidepressants and amphetamine block the effects of 6-OHDA on DA neurons, and a dat-1 deletion line (gift of J. Rand) is insensitive to the neurotoxin. Ultrastructural analysis of the worm DA neurons shows significant signs of neurodegeneration including small, dark, and rounded cell bodies, as well as vacuolation and loss of **neuronal** processes following 6-OHDA exposure. Although these phenotypes are characteristic of apoptosis both in mammals and worms, cell death appears to be independent of the classic apoptotic pathway, since the caspase-deficient ced-3 and ced-4 backgrounds still display the toxin-induced **neuronal** blebbing and loss of GFP expression. These studies as well as our progress on toxin-based genetic **screens** for regulators of DAT-1 and toxin-mediated cell death will be presented.

L16 ANSWER 12 OF 27 MEDLINE on STN
 ACCESSION NUMBER: 2001158845 MEDLINE
 DOCUMENT NUMBER: 21103917 PubMed ID: 11181837
 TITLE: A cGMP-dependent protein kinase is implicated in wild-type motility in *C. elegans*.
 AUTHOR: Stansberry J; Baude E J; Taylor M K; Chen P J; Jin S W; Ellis R E; Uhler M D
 CORPORATE SOURCE: Department of Biological Chemistry, Neuroscience Graduate Program, University of Michigan, Ann Arbor, Michigan, USA.
 CONTRACT NUMBER: GM50791 (NIGMS)
 SOURCE: JOURNAL OF NEUROCHEMISTRY, (2001 Feb) 76 (4) 1177-87. Journal code: 2985190R. ISSN: 0022-3042.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200103
 ENTRY DATE: Entered STN: 20010404
 Last Updated on STN: 20010404
 Entered Medline: 20010322

AB In mammals, cyclic GMP and cGMP-dependent protein kinases (cGKs) have been implicated in the regulation of many neuronal functions including long-term potentiation and long-term depression of synaptic efficacy. To develop *Caenorhabditis elegans* as a model system for studying the neuronal function of the cGKs, we cloned and characterized the *cgk-1* gene. A combination of approaches showed that *cgk-1* produces three transcripts, which differ in their first exon but are similar in length. Northern analysis of *C. elegans* RNA, performed with a probe designed to hybridize to all three transcripts, confirmed that a major 3.0 kb *cgk-1* transcript is present at all stages of development. To determine if the CGK-1C protein was a cGMP-dependent protein kinase, CGK-1C was expressed in SF:9 cells and purified. CGK-1C shows a $K(a)$ of 190 +/- 14 nM for cGMP and 18.4 +/- 2 microM for cAMP. Furthermore, CGK-1C undergoes autophosphorylation in a cGMP-dependent manner and is inhibited by the commonly used cGK inhibitor, KT5823. To determine which cells expressed CGK-1C, a 2.4-kb DNA fragment from the promoter of CGK-1C was used to drive GFP expression. The CGK-1C reporter construct is strongly expressed in the ventral nerve cord and in several other neurons as well as the marginal cells of the pharynx and intestine. Finally, RNA-mediated interference of CGK-1 resulted in movement defects in nematode larvae. These results provide the first demonstration that cGMP-dependent protein kinase is present in neurons of *C. elegans* and show that this kinase is required for normal motility.

16 ANSWER 17 OF 27 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN

ACCESSION NUMBER: 2001:55358 BIOSIS

DOCUMENT NUMBER: PREV200100055358

TITLE: IDA-1, a *Caenorhabditis elegans* homolog of the diabetic autoantigens IA-2 and phogrin, is expressed in peptidergic neurons in the worm.

AUTHOR(S): Zahn, Tobias R.; Macmorris, Margaret A.; Dong, Weijia; Day, Robert; Hutton, John C. [Reprint author]

CORPORATE SOURCE: Barbara Davis Center for Childhood Diabetes, University of Colorado Health Sciences Center, 4200 East 9th Avenue, Denver, CO, 80262, USA
john.hutton@uchsc.edu

SOURCE: Journal of Comparative Neurology, (January 1, 2001) Vol. 429, No. 1, pp. 127-143. print.
CODEN: JCNEAM. ISSN: 0021-9967.

DOCUMENT TYPE: Article

LANGUAGE: English

OTHER SOURCE: Genbank-AJ245560

ENTRY DATE: Entered STN: 24 Jan 2001

Last Updated on STN: 15 Feb 2002

AB The closely related mammalian proteins IA-2 and phogrin are protein tyrosine phosphatase-like receptor proteins spanning the membrane of dense core vesicles of neuroendocrine tissues. They are of interest as molecular components of the secretory machinery and as major targets of autoimmunity in type I diabetes mellitus. The *Caenorhabditis elegans* genome has a single copy of an IA-2/phogrin homolog *ida-1* III (islet cell diabetic autoantigen), which encodes the *ida-1* (B0244.2) gene product as a series of 12 exons over a 10-kb region of chromosome III. The full-length sequence of the *ida-1* cDNA encoded a 767-amino acid type 1 transmembrane protein of 87 kDa. The PTP catalytic site consensus sequence of IDA-1, like IA-2 and phogrin, diverged and would not be active. Expression of green fluorescent protein (GFP) under the *ida-1* gene promoter showed activity in a subset of around 30 neurons with sensory functions and the uvl cells of the vulva in hermaphrodites. Males showed additional expression in male-specific neurons. In situ experiments in rat brain showing the distribution of IA-2 and phogrin suggested a complimentary and overlapping pattern compared with the proprotein convertases PC1 and PC2. In *C. elegans*, IDA-1-expressing cells comprised a subset of those expressing the PC2 homolog KPC-2 (C51E3.7), consistent with IDA-1 being a component of neuropeptide-containing dense core vesicles. The results support the hypothesis that *C. elegans* IDA-1 is the functional homolog of IA-2 and phogrin in mammals. Analysis of the function of IDA-1 should contribute to our understanding of the function of these proteins in signal transduction, vesicle locomotion, and exocytosis.

L16 ANSWER 20 OF 27 MEDLINE on STN
 ACCESSION NUMBER: 2001182549 MEDLINE
 DOCUMENT NUMBER: 21100414 PubMed ID: 11167007
 TITLE: Two isoforms of sarco/endoplasmic reticulum calcium ATPase (SERCA) are essential in *Caenorhabditis elegans*.
 AUTHOR: Cho J H; Bandyopadhyay J; Lee J; Park C S; Ahnn J
 CORPORATE SOURCE: Department of Life Science, Kwangju Institute of Science and Technology, 500-712, Kwangju, South Korea..
 joohong@eunhasu.kjist.ac.kr
 SOURCE: GENE, (2000 Dec 31) 261 (2) 211-9.
 Journal code: 7706761. ISSN: 0378-1119.
 PUB. COUNTRY: Netherlands
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200103
 ENTRY DATE: Entered STN: 20010404
 Last Updated on STN: 20010404
 Entered Medline: 20010329

AB SERCA (Sarco/Endoplasmic Reticulum Calcium ATPase), a membrane bound $\text{Ca}(2+)\text{-}/\text{Mg}(2+)\text{-}$ dependent ATPase that sequesters $\text{Ca}(2+)$ into the SR/ER lumen, is one of the essential components for the maintenance of intracellular $\text{Ca}(2+)$ homeostasis. Here we describe the identification and functional characterization of a *C. elegans* SERCA gene (*ser-1*). *ser-1* is a single gene alternatively spliced at its carboxyl terminus to form two isoforms (SER-1A and SER-1B) and displays a high homology (70% identity, 80% similarity) with mammalian SERCAs. Green fluorescent protein (GFP) and whole-mount immunostaining analyses reveal that SER-1 expresses in neuronal cells, body-wall muscles, pharyngeal and vulval muscles, excretory cells, and vulva epithelial cells. Furthermore, SER-1::GFP expresses during embryonic stages and the expression is maintained through the adult stages. Double-stranded RNA injection (also known as RNAi) targeted to each SER-1 isoform results in severe phenotypic defects: *ser-1A*(RNAi) animals show embryonic lethality, whereas *ser-1B*(RNAi) results in L1 larval arrest phenotype. These findings suggest that both isoforms of *C. elegans* SERCA, like in mammals, are essential for embryonic development and post-embryonic growth and survival.